

(FILE 'HOME' ENTERED AT 17:37:50 ON 20 JUN 2000)

FILE 'MEDLINE, BIOSIS, CAPLUS, USPATFULL' ENTERED AT 17:38:38 ON 20 JUN 2000

L1 8621 S NEUROECTODERM?
L2 1342108 S ANTIBODY OR ANTIBODIES
L3 56 S CHLOROTOXIN?
L4 8267 S CHLORIDE CHANNEL
L5 2525715 S TUMOR? OR CANCER? OR NEOPLAS?
L6 20 S L3 AND L4
L7 454 S L2 AND L4
L8 9 S L6 AND L5
L9 108 S L7 AND L5

=> dup rem l8

PROCESSING COMPLETED FOR L8

L10 6 DUP REM L8 (3 DUPLICATES REMOVED)

=> d l10 1-6 bib abs

L10 ANSWER 1 OF 6 USPATFULL
AN 2000:21668 USPATFULL
TI Method of diagnosing and treating gliomas
IN Ullrich, Nicole, Fairfield, CT, United States
Sontheimer, Harald W., Birmingham, AL, United States
PA UAB Research Foundation, Birmingham, AL, United States (U.S.
corporation)
PI US 6028174 20000222
AI US 1997-980388 19971128 (8)
RLI Division of Ser. No. US 1996-774154, filed on 26 Dec 1996
PRAI US 1995-9283 19951227 (60)
DT Utility
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Sun-Hoffman,
Lin
LREP Adler, Benjamin Aaron
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN 20 Drawing Figure(s); 20 Drawing Page(s)
LN.CNT 1434
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a recombinant toxin and monoclonal
antibody which specifically binds to glial-derived or
meningioma-derived
tumor cells. Also provided are various methods of screening for
malignant gliomas and meningiomas. Further provided are methods of
treating malignant gliomas, including glioblastoma multiforme and
astrocytomas.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 1
AN 1999:330028 CAPLUS
DN 130:335024
TI Method of diagnosing and treating gliomas
IN Ullrich, Nicole; Sontheimer, Harald W.
PA UAB Research Foundation, USA
SO U.S., 34 pp.

CODEN: USXXAM

DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5905027	A	19990518	US 1996-774154	19961226
	US 6028174	A	20000222	US 1997-980388	19971128
PRAI	US 1995-9283		19951227		
	US 1996-774154		19961226		

AB The present invention provides a recombinant toxin and monoclonal antibody which specifically binds to glial-derived or meningioma-derived **tumor** cells. Also provided are various methods of screening for malignant gliomas and meningiomas. Further provided are methods of treating malignant gliomas, including glioblastoma multiforme and astrocytomas.

RE.CNT 2
RE

- (1) Ullrich; Am J Physiol 1996, V270(5, pt 1), PC1511
- (2) Ullrich; Neuro Report 1996, V7(5), P1020 MEDLINE

L10 ANSWER 3 OF 6 MEDLINE DUPLICATE 2
AN 1999337948 MEDLINE
DN 99337948
TI Modulation of glioma cell migration and invasion using Cl(-) and K(+) ion channel blockers.
AU Soroceanu L; Manning T J Jr; Sontheimer H
CS Department of Neurobiology, The University of Alabama at Birmingham, Birmingham, Alabama 35294-0021, USA.
NC NS36692 (NINDS)
SO JOURNAL OF NEUROSCIENCE, (1999 Jul 15) 19 (14) 5942-54.
Journal code: JDF. ISSN: 0270-6474.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
EW 19991001
AB Human malignant gliomas are highly invasive **tumors**. Mechanisms that allow glioma cells to disseminate, migrating through the narrow extracellular brain spaces are poorly understood. We recently demonstrated expression of large voltage-dependent chloride (Cl(-)) currents, selectively expressed by human glioma cells in vitro and in situ (Ullrich et al., 1998). Currents are sensitive to several Cl(-) channel blockers, including **chlorotoxin** (Ctx), (Ullrich and Sontheimer; 1996; Ullrich et al; 1996), tetraethylammonium chloride (TEA), and tamoxifen (Ransom and Sontheimer, 1998). Using Transwell migration assays, we show that blockade of glioma Cl(-) channels specifically inhibits **tumor** cell migration in a dose-dependent manner. Ctx (5 microM), tamoxifen (10 microM), and TEA (1 mM) also prevented invasion of human glioma cells into fetal rat brain aggregates, used as an in vitro model to assess **tumor** invasiveness. Anion replacement studies suggest that permeation of chloride ions through glioma **chloride channel** is obligatory for cell migration. Osmotically induced cell swelling and subsequent regulatory volume decrease (RVD) in cultured glioma cells were reversibly prevented by 1 mM TEA, 10 microM tamoxifen, and irreversibly blocked by 5 microM Ctx added to the hypotonic media. Cl(-) fluxes associated with adaptive shape changes elicited by cell swelling and RVD in glioma cells were inhibited by 5 microM Ctx, 10 microM tamoxifen, and 1 mM TEA, as determined using the Cl(-)-sensitive fluorescent dye 6-methoxy-N-ethylquinolinium iodide. Collectively, these

data suggest that chloride channels in glioma cells may enable **tumor** invasiveness, presumably by facilitating cell shape and cell volume changes that are more conducive to migration and invasion.

L10 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2000 ACS

AN 1998:111924 CAPLUS

DN 128:229085

TI Expression of voltage-activated chloride currents in acute slices of human

gliomas

AU Ullrich, N.; Bordey, A.; Gillespie, G. Y.; Sontheimer, H.

CS Department of Neurobiology, University of Alabama at Birmingham, Birmingham, AL, 35294, USA

SO Neuroscience (Oxford) (1998), 83(4), 1161-1173

CODEN: NRSCDN; ISSN: 0306-4522

PB Elsevier Science Ltd.

DT Journal

LA English

AB Using whole-cell patch-clamp recordings, we identified a novel voltage-activated chloride current that was selectively expressed in glioma cells from 23 patient biopsies. Chloride currents were identified in 64% of glioma cells studied in acute slices of nine patient biopsies. These derived from gliomas of various pathol. grades. In addn., 98% of cells acutely isolated or in short-term culture from 23 patients diagnosed

with gliomas showed chloride current expression. These currents, which we

termed glioma chloride currents activated at potentials >45 mV, showed pronounced outward rectification, and were sensitive to bath application of the presumed Cl⁻ channel specific peptide **chlorotoxin** (.apprx.600 nM) derived from Leiurus scorpion venom. Interestingly, low grade **tumors** (e.g., pilocytic astrocytomas), contg. more differentiated, astrocyte-like cells showed expression of glioma chloride currents in concert with voltage-activated sodium and potassium currents also seen in normal astrocytes. By contrast, high grade **tumors** (e.g., glioblastoma multiforme) expressed almost exclusively chloride currents, suggesting a gradual loss of Na⁺ currents and gain of Cl⁻ currents with increasing pathol. **tumor** grade. To expand on the observation that these chloride currents are glioma-specific, we introduced exptl. **tumors** in scid mice by intracranial injection of D54MG glioma cells and subsequently recorded from **tumor** cells and adjacent normal glial cells in acute slices. We consistently obsd. expression of **chlorotoxin**-sensitive chloride channels in implanted glioma cells, but without evidence for expression of chloride channels in surrounding "normal" host glial cells, suggesting that these chloride channels are probably a glioma-specific feature. Finding of

this novel glioma specific Cl⁻ channel in gliomas in situ and it's selective binding of **chlorotoxin** may provide a way to identify or target glioma cells in the future.

L10 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2000 ACS

AN 1997:505749 CAPLUS

DN 127:119322

TI Method of diagnosing and treating gliomas

IN Sontheimer, Harald W.; Ullrich, Nicole

PA UAB Research Foundation, USA

SO PCT Int. Appl., 81 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	WO 9724619	A1	19970710	WO 1996-US20403	19961227

W: AU, CA, JP
 RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT,
 SE
 CA 2249351 AA 19970710 CA 1996-2249351 19961227
 AU 9722399 A1 19970728 AU 1997-22399 19961227
 EP 953153 A1 19991103 EP 1996-946129 19961227
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, FI
 PRAI US 1995-9283 19951227
 WO 1996-US20403 19961227

AB The present invention relates generally to the fields of cell physiol.,
 neurol. and neuro-oncol. More specifically, the present invention
 relates

to a novel method of detection of the membrane protein "glioma
chloride channel" for use as a specific **tumor**
 marker for the diagnosis and treatment of gliomas and meningiomas. The
 invention describes the expression of this chloride conductance with
 unique properties that selectively characterizes **tumor**-derived
 cells of glial origin. Whole-cell patch-clamp techniques were used to
 characterize the biophys. and pharmacol. properties of chloride channels
 in primary cultures and acutely isolated cells from biopsies of human
 astrocytomas and established cell lines.

L10 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2000 ACS

AN 1996:325861 CAPLUS

DN 125:48543

TI Biophysical and pharmacological characterization of chloride currents in
 human astrocytoma cells

AU Ullrich, Nicole; Sontheimer, Harald

CS Neurobiology Research Center, University Alabama Birmingham, Birmingham,
 AL, 35294, USA

SO Am. J. Physiol. (1996), 270(5, Pt. 1), C1511-C1521

CODEN: AJPHAP; ISSN: 0002-9513

DT Journal

LA English

AB Expression of voltage-activated ion channels was studied in primary
 cultures from seven freshly resected human primary brain **tumors**
 and in an established human astrocytoma cell line, STTG1. Astrocytoma
 cells consistently expressed voltage-dependent outwardly rectifying
 currents. Currents activated at potentials >45 mV and showed outward
 transients on termination of voltage steps. Currents reversed at the Cl-
 equil. potential, suggesting that they were largely carried by Cl-.
 Altering extracellular K+ or Na+ concn. did not alter currents; neither
 did replacement of intracellular K+ by Cs+ or intracellular Na+ by
 N-methyl-D-glucosamine. Anion-substitution expts. suggest the following
 permeability sequence, detd. from shifts in tail current reversal
 potential: I- > NO3- > Br- > Cl- > acetate > isethionate > F- >
 glutamate.

Currents were sensitive to the Cl- channel blockers **chlorotoxin**,
 4,4'-diisothiocyanostilbene-2,2'-disulfonic acid (DIDS), and
 4,4'-dinitrostilbene-2,2'-disulfonic acid (DNDS), with **chlorotoxin**
 being most effective, yielding >80% block at 590 nM. DIDS (100 .mu.M)

and

DNDS (100 .mu.M) reduced currents by 33.5 and 38.2%, resp. Currents were
 also sensitive to Zn2+ (100 .mu.M, 47% block) and Cd2+ (25 .mu.M, 42%
 block). Reducing extracellular Ca2+ concn. decreased outward currents by
 58% and almost completely eliminated transients, suggesting that Cl-
 currents are Ca2+ dependent. Cl- channel block resulted in altered cell
 proliferation as detd. by [3H]thymidine incorporation, suggesting that

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L9 108 S L7 AND L5
L10 6 DUP REM L8 (3 DUPLICATES REMOVED)

=> s l9 and l1

L11 3 L9 AND L1

=> dup rem l11

PROCESSING COMPLETED FOR L11

L12 3 DUP REM L11 (0 DUPLICATES REMOVED)

=> d l12 1-3 bib abs

L12 ANSWER 1 OF 3 USPATFULL

AN 2000:37945 USPATFULL

TI Hydroxy and ether-containing oxyalkylene esters and uses thereof

IN Nudelman, Abraham, Rehovot, Israel

Rephaeli, Ada, North Caldwell, NJ, United States

PA Mor Research Applications, Ltd., Givat Shmuel, Israel (non-U.S. corporation)

Beacon Laboratories, Inc., Phoenix, MD, United States (U.S. corporation)

Bar-Ilan University, Ramat-Gan, Israel (non-U.S. corporation)

PI US 6043389 20000328

AI US 1997-814224 19970311 (8)

DT Utility

EXNAM Primary Examiner: Reamer, James H.

LREP Kenyon & Kenyon

CLMN Number of Claims: 4

ECL Exemplary Claim: 1

DRWN 4 Drawing Figure(s); 2 Drawing Page(s)

LN.CNT 1117

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to compositions for and methods of treating, preventing or ameliorating **cancer** and other proliferative diseases as well as methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, inhibiting telomerase activity, treating virus-associated **tumors**, especially EBV-associated **tumors**, augmenting expression of **tumor** suppressor genes, inducing tolerance to antigens, or treating, preventing or

ameliorating protozoan infection or inhibiting histone deacetylase in cells. The compositions of the invention are to and the methods of the invention use hydroxy and ether-containing oxyalkylene esters.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 2 OF 3 USPATFULL
AN 2000:24635 USPATFULL
TI Oxyalkylene phosphate compounds and uses thereof
IN Nudelman, Abraham, Rehovot, Israel
Rephaeli, Ada, North Caldwell, NJ, United States
PA Bar-Ilan Research & Development Co., Ltd., Ramat-Gan, Israel (non-U.S. corporation)
Mor Research Applications Ltd., Givat Shmuel, Israel (non-U.S. corporation)
PI US 6030961 20000229
AI US 1997-814386 19970311 (8)
DT Utility
EXNAM Primary Examiner: Raymond, Richard L.
LREP Kenyon & Kenyon
CLMN Number of Claims: 52
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1482

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to compositions for and methods of treating, preventing or ameliorating **cancer** and other proliferative diseases as well as methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, inhibiting telomerase activity, treating virus-associated **tumors**, especially EBV-associated **tumors**, modulating gene expression and in particular, augmenting expression of **tumor** suppressor genes, inducing tolerance to antigens, treating, preventing or ameliorating protozoan infection, or inhibiting histone deacetylase in cells. The compositions of the invention are to and the methods of the invention use oxyalkylene phosphate compounds.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 3 OF 3 USPATFULL
AN 1999:96406 USPATFULL
TI Therapeutic augmentation of oxyalkylene diesters and butyric acid derivatives
IN Rephaeli, Ada, North Caldwell, NJ, United States
PA Beacon Laboratories, Inc., Phoenix, MD, United States (U.S. corporation)
PI US 5939455 19990817
AI US 1997-814222 19970311 (8)
DT Utility
EXNAM Primary Examiner: Weddington, Kevin E.
LREP Kenyon & Kenyon
CLMN Number of Claims: 63
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1545

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides a method of augmenting the therapeutic activity of an oxyalkylene-containing compound, butyric acid, a butyric acid salt or butyric acid derivative by administering an inhibitor of .beta.-oxidation of fatty acids to a patient or to host cells. Pharmaceutical compositions are also included.

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L11 3 S L9 AND L1
L12 3 DUP REM L11 (0 DUPLICATES REMOVED)

=> s l1 and l2 and l4

L13 3 L1 AND L2 AND L4

=> d l13 1-3 bib abs kwic

L13 ANSWER 1 OF 3 USPATFULL
AN 2000:37945 USPATFULL
TI Hydroxy and ether-containing oxyalkylene esters and uses thereof
IN Nudelman, Abraham, Rehovot, Israel
Rephaeli, Ada, North Caldwell, NJ, United States
PA Mor Research Applications, Ltd., Givat Shmuel, Israel (non-U.S. corporation)
Beacon Laboratories, Inc., Phoenix, MD, United States (U.S. corporation)
Bar-Ilan University, Ramat-Gan, Israel (non-U.S. corporation)
PI US 6043389 20000328
AI US 1997-814224 19970311 (8)
DT Utility
EXNAM Primary Examiner: Reamer, James H.
LREP Kenyon & Kenyon
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1117
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to compositions for and methods of treating, preventing or ameliorating cancer and other proliferative diseases as well as methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, inhibiting telomerase activity, treating virus-associated tumors, especially EBV-associated tumors, augmenting expression of tumor suppressor genes, inducing tolerance to antigens, or treating, preventing or ameliorating protozoan infection or inhibiting histone deacetylase in cells. The compositions of the invention are to and the methods of the invention use hydroxy and ether-containing oxyalkylene esters.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . ovarian cancers, pancreatic cancers, hepatocarcinomas, prostate cancers, squamous carcinomas, other dermatologic malignancies, teratocarcinomas, T-cell lymphomas, lung tumors, gliomas, neuroblastomas, peripheral **neuroectodermal** tumors, rhabdomyosarcomas, and prostate tumors and other solid tumors. It is also possible that compounds of Formula I have anti-proliferative. .

SUMM . . . are not limited to, thalassemias, sickle cell anemias, infectious anemias, aplastic anemias, hypoplastic and hypoproliferative anemias, sideroblastic anemias, myelophthisic anemias, **antibody**-mediated anemias, anemias due to chronic diseases and enzyme-deficiencies, and anemias due to blood loss, radiation therapy and chemotherapy. In this. . .

SUMM . . . agents of the invention for the above method include, but are not limited to, cytokines, interleukins, anti-cancer agents, chemotherapeutic agents, **antibodies**, conjugated **antibodies**, immune stimulants, antibiotics, hormone antagonists, and growth stimulants. The compounds of the invention can be administered prior to, after or. . .

SUMM . . . or ameliorating symptoms in cystic fibrosis patients by administering an amount of a compound of Formula I effective to enhance **chloride channel** expression.

DETD . . . recombinant gene expression; to modulate gene expression; to augment expression of tumor suppressor genes; to enhance insulin expression; to enhance **chloride channel** expression, to induce tolerance to an antigen; to treat, prevent or ameliorate protozoan infection; or to inhibit histone deacetylase in. . .

DETD . . . differentiating agents. For example, the pharmaceutical agent can be a cytokine, an interleukin, an anti-cancer agent, a chemotherapeutic agent, an **antibody**, a conjugated **antibody**, an immune stimulant, an antibiotic, a hormone antagonist or a growth stimulant. The pharmaceutical agent can also be a cytotoxic. . .

DETD . . . al., Md. Carcin. 3:350-362, 1990). Casein detection can be done by histochemical staining of breast cancer cells using a human **antibody** to human casein as described by Cheung et al., J. Clin. Invest. 75:1722-1728, which is incorporated by reference in its. . .

DETD . . . prepared and the cells are fixed with ethanol. Fixed cells are reacted overnight at 4.degree. C. with the primary monoclonal **antibody**, anti-Bcl-2 at a dilution of 1:50. Staining is completed to visualize **antibody** binding, for example, using Strep A-B Universal Kit (Sigma) in accordance with the manufacturer's instructions. Identically-treated cells which received no primary **antibody** can serve as a non-specific binding control. Commercial kits are also available and can be used for detecting apoptosis, for.

DETD . The level of CD11b was measured on HL-60 cells by flow cytometry using a monoclonal **antibody** (MAb) against CD11b in a standard indirect immunofluorescence assay. Cells were cultured for 6 days with the indicated concentration of. . .

L13 ANSWER 2 OF 3 USPATFULL
AN 2000:24635 USPATFULL
TI Oxyalkylene phosphate compounds and uses thereof
IN Nudelman, Abraham, Rehovot, Israel
Rephaeli, Ada, North Caldwell, NJ, United States
PA Bar-Ilan Research & Development Co., Ltd., Ramat-Gan, Israel (non-U.S. corporation)
Mor Research Applications Ltd., Givat Shmuel, Israel (non-U.S.

corporation)
PI US 6030961 20000229
AI US 1997-814386 19970311 (8)
DT Utility
EXNAM Primary Examiner: Raymond, Richard L.
LREP Kenyon & Kenyon
CLMN Number of Claims: 52
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1482

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to compositions for and methods of treating, preventing or ameliorating cancer and other proliferative diseases as well as methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, inhibiting telomerase activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and in particular, augmenting expression of tumor suppressor genes, inducing tolerance to antigens, treating, preventing or ameliorating protozoan infection, or inhibiting histone deacetylase in cells. The compositions of the invention are to and the methods of the invention use oxyalkalene phosphate compounds.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . ovarian cancers, pancreatic cancers, hepatocarcinomas, prostate

cancers, squamous carcinomas, other dermatologic malignancies, teratocarcinomas, T-cell lymphomas, lung tumors, gliomas, neuroblastomas, peripheral **neuroectodermal** tumors, rhabdomyosarcomas, and prostate tumors and other solid tumors. It is also possible that compounds of Formula I as defined. . .

SUMM . . . are not limited to, thalassemias, sickle cell anemias, infectious anemias, aplastic anemias, hypoplastic and hypoproliferative anemias, sideroblastic anemias, myelophthisic anemias, **antibody**-mediated anemias, anemias due to chronic diseases and enzyme-deficiencies, and anemias due to blood loss, radiation therapy and chemotherapy. In this. . .

SUMM . . . agents of the invention for the above method include but are not limited to, cytokines, interleukins, anti-cancer agents, chemotherapeutic agents, **antibodies**, conjugated **antibodies**, immune stimulants, antibiotics, hormone antagonists, and growth stimulants. The compounds of the invention can be administered prior to, after or. . .

SUMM . . . in cystic fibrosis patients by administering an amount of a compound of Formula I as defined above effective to enhance **chloride channel** expression.

DETD . . . immune response; to enhance gene expression; modulate or augment expression of tumor suppressor genes; to enhance insulin expression; to enhance **chloride channel** expression; to induce tolerance to an antigen; to treat, prevent or ameliorate protozoan infection; or to inhibit histone deacetylase in. . .

DETD . . . differentiating agents. For example, the pharmaceutical agent can be a cytokine, an interleukin, an anti-cancer agent, a chemotherapeutic agent, an **antibody**, a conjugated **antibody**, an immune stimulant, an antibiotic, a hormone antagonist or a growth stimulant. The pharmaceutical agent can also be

a

cytotoxic. . .

DETD . . . al., Md. Carcin. 3:350-362, 1990). Casein detection can be done

by histochemical staining of breast cancer cells using a human **antibody** to human casein as described by Cheung et al., J. Clin.

Invest. 75:1722-1728, which is incorporated by reference in its. . .

DETD . . . prepared and the cells are fixed with ethanol. Fixed cells are reacted overnight at 4.degree. C. with the primary monoclonal **antibody**, anti-Bcl-2 at a dilution of 1:50. Staining is completed to visualize **antibody** binding, for example, using Strep A-B Universal Kit (Sigma) in accordance with the manufacturer's instructions. Identically-treated cells which received no primary **antibody** can serve as a non-specific binding control. Commercial kits are also available and can be used for detecting apoptosis, for.

DETD The level of CD11b was measured on HL-60 cells by flow cytometry using a monoclonal **antibody** (MAb) against CD11b in a standard indirect immunofluorescence assay. Cells were cultured for three or six days with the indicated. . .

CLM What is claimed is:
. . . selected from the group consisting of a cytokine, an interleukin, an anti-cancer agent of anti-neoplastic agent, a chemotherapeutic agent, an **antibody**, a conjugated **antibody**, an immune stimulant, antibiotic, a hormone antagonist and a growth stimulant.

L13 ANSWER 3 OF 3 USPATFULL

AN 1999:96406 USPATFULL

TI Therapeutic augmentation of oxyalkylene diesters and butyric acid derivatives

IN Rephaeli, Ada, North Caldwell, NJ, United States

PA Beacon Laboratories, Inc., Phoenix, MD, United States (U.S. corporation)

PI US 5939455 19990817

AI US 1997-814222 19970311 (8)

DT Utility

EXNAM Primary Examiner: Weddington, Kevin E.

LREP Kenyon & Kenyon

CLMN Number of Claims: 63

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 1545

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides a method of augmenting the therapeutic activity of an oxyalkylene-containing compound, butyric acid, a butyric acid salt or butyric acid derivative by administering an inhibitor of .beta.-oxidation of fatty acids to a patient or to host cells. Pharmaceutical compositions are also included.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . pharmaceutical compositions further containing a pharmaceutical

agent selected from a cytokine, an interleukin, an anti-cancer agent, a chemotherapeutic agent, an **antibody**, a conjugated **antibody**, an immune stimulant, an antibiotic, a hormone antagonist, a growth stimulant, an antiviral agent or a cytotoxic agent.

DETD . . . ovarian cancers, pancreatic cancers, hepatocarcinomas, prostate cancers, squamous carcinomas, other dermatologic malignancies, teratocarcinomas, T-cell lymphomas, lung tumors, gliomas, neuroblastomas, peripheral **neuroectodermal** tumors, rhabdomyosarcomas, and prostate tumors and other solid tumors. It is also possible that compounds of the invention have anti-proliferative.

DETD . . . agents of the invention for the above method include, but are not limited to, cytokines, interleukins, anti-cancer agents, chemotherapeutic agents, **antibodies**, conjugated **antibodies**, immune stimulants, antibiotics, hormone antagonists, and growth stimulants. The .beta.-oxidation inhibitor and compounds of the invention can be administered prior. . . .

DETD . . . are not limited to, thalassemias, sickle cell anemias, infectious anemias, aplastic anemias, hypoplastic and hypoproliferative anemias, sideroblastic anemias, myelophthisic anemias, **antibody**-mediated anemias, anemias due to chronic diseases and enzyme-deficiencies, and anemias due to blood loss, radiation therapy and chemotherapy. In this. . . .

DETD In yet another embodiment, the therapeutic activity is effective to enhance **chloride channel** expression in a cystic fibrosis patient.

DETD . . . recombinant gene expression; to modulate gene expression; to augment expression of tumor suppressor genes; to enhance insulin expression; to enhance **chloride channel** expression; to induce tolerance to an antigen; to treat, prevent or ameliorate protozoan infection; or to inhibit histone deacetylase in. . . .

DETD . . . as differentiating agents. Further, the pharmaceutical agent can be a cytokine, an interleukin, an anti-cancer agent, a chemotherapeutic agent, an **antibody**, a conjugated **antibody**, an immune stimulant, an antibiotic, a hormone antagonist or a growth stimulant. The pharmaceutical agent can also be a

cytotoxic. . . .

DETD . . . al., Md. Carcin. 3:350-362, 1990). Casein detection can be done by histochemical staining of breast cancer cells using a human **antibody** to human casein as described by Cheung et al., J. Clin. Invest. 75:1722-1728, which is incorporated by reference in its. . . .

DETD . . . prepared and the cells are fixed with ethanol. Fixed cells are reacted overnight at 4.degree. C. with the primary monoclonal **antibody**, anti-Bcl-2 at a dilution of 1:50. Staining is completed to visualize **antibody** binding, for example, using Strep A-B Universal Kit (Sigma) in accordance with the manufacturer's instructions. Identically-treated cells which received no primary **antibody** can serve as a non-specific binding control. Commercial kits are also available and can be used for detecting apoptosis, for.

CLM What is claimed is:

. . . from the group consisting of a cytokine, an interleukin, an anti-cancer agent or an anti-neoplastic agent, a chemotherapeutic agent,

an **antibody**, a conjugated **antibody**, an immune stimulant, an antibiotic, a hormone antagonist or a growth stimulant.

36. The method of claim 1 wherein said therapeutic activity is effective to enhance **chloride channel** expression in a cystic fibrosis patient.

. . . a pharmaceutical agent selected from the group consisting of a cytokine, an interleukin, an anti-cancer agent, a chemotherapeutic agent, an **antibody**, a conjugated **antibody**, an immune stimulant, an antibiotic, a hormone antagonist, a growth stimulant, an antiviral agent and a cytotoxic agent.